Application of Experimental Results.

If the thesis as to danger ratio is established, when we wish to apply our animal experiments in the clinic it seems clear that we must know approximately what is the ratio for a given drug with the higher mammals. Further, an anaesthetic with a low danger ratio is much safer for trial than one with a high, the risk of incautiously approaching the lethal dose being less.

Butyn.³⁸

Experimentally butyn appears, in the hands of four independent workers, to be of toxicity equal to cocaine or rather more toxic. Many of the experiments were done with strong solutions, and there seems some evidence that with weak solutions the toxicity of butyn is less reduced than that of cocaine—that is, its relative toxicity is greater. It is said to have special advantages in eye work.³⁹ In the nose it seems an effective anaesthetic, but more irritant than cocaine.⁴⁰ By courtesy of the proprietors I have received notes of five deaths following its clinical applications: two for septal resection—5 per cent. solution on tampon; two for tonsil operation*—1/4 per cent. solution injected—no evidence of overdose; one case of definite overdose. Obviously considerable caution is required in using it.

Note on Atoxodine.

The claim of this anaesthetic to be without toxicity was found to have no foundation: the toxicity is low. It appears useless on mucous surfaces.

Summary.

1. The toxicity of various local anaesthetics was compared experimentally by determining the minimal lethal doses.
2. The effect of dilution was found to be a lowering of toxicity.
3. Addition of adrenaline definitely lowered toxicity.
4. It appears that the dose of anaesthetic which will produce alarming symptoms is with different drugs a different fraction of the M.L.D., cocaine having a small fraction, for example, butyn a larger.
5. Caution is required in using butyn.

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* Cocaine was painted on fauces in one case.

A British Medical Association Lecture

ON

THE PATHOLOGY OF GASTRIC ULCER.

DELIVERED TO THE DERBY DIVISION, MAY 18TH, 1923,

BY

MATTHEW J. STEWART, M.B., CH.B., M.R.C.P.,

PROFESSOR OF PATHOLOGY, UNIVERSITY OF LEEDS.

(Continued from page 957.)

PART II.—PATHOLOGICAL OBSERVATIONS ON GASTRIC AND DUODENAL ULCER.

THE statistical data utilized in this section were obtained from two sources: (a) a consecutive series of 6,800 autopsies at the Leeds General Infirmary, of which a special group of 1,500 were intensively studied from the point of view of gastro-duodenal pathology, and (b) a series of 120 stomach specimens removed at operation and submitted to microscopic examination.

A. INCIDENCE OF PEPTIC ULCER.

In the intensively studied series of 1,500 autopsies there were 130 cases of gastro-duodenal ulceration (that is, 8.7 per cent.) equally distributed between the two organs (4.5 per cent. in each). In 28 cases (1.9 per cent.) chronic gastric ulcers were present, and in 58 (3.9 per cent.) chronic duodenal. In the full series of 6,800 autopsies there were 150 cases with chronic gastric ulcer (2.2 per cent.) and 200 with chronic duodenal ulcer (2.9 per cent.). The relative frequency here shown (3:4) differs considerably from that revealed by clinical statistics. Sir Berkeley Moynihan's (1923)²³ latest review of 718 cases coming to operation shows duodenal ulcer to be three times more frequent than gastric, while in the Mayo Clinic figures (C. H. Mayo, 1920)²⁹ for 5,723 cases the ratio is practically 4:1. This discrepancy between autopsy and clinical statistics may be due to the surgeon seeing a relatively larger proportion of duodenal ulcer cases, which would suggest that, symptomatically, this is the more severe disease, or it may be due to the fact that gastric ulcer is the more difficult to diagnose, with the result that fewer cases come to operation. On the other hand, the partiality may lie with the autopsy figures. Gastric ulcer is the more serious and more fatal disease, with a much heavier operation mortality; a larger proportion of the duodenal cases, therefore, will recover.

The scars of healed ulcers are met with fairly frequently. In the series of 1,500 autopsies, 42 cases (2.8 per cent.) showed gastric scars, and 36 cases (2.4 per cent.) duodenal. Coincident chronic lesions (ulcers or scars) of the stomach and duodenum were met with in 7 cases, or 0.45 per cent. of the whole, but in only a single instance did an open chronic ulcer coexist in the two organs.

B. AGE AND SEX INCIDENCE.

Acute ulcer of the stomach and duodenum occurs with a fairly uniform frequency throughout the decades, except that it is rather less common under 20. Chronic ulcer is rare under 20, only 1.5 per cent. of all cases coming to autopsy in the first two decades. In the present series 90 per cent. of cases of chronic gastric ulcer came to autopsy between the ages of 30 and 69, while the corresponding figures for chronic duodenal ulcer and cancer of the stomach were 84 per cent. and 96 per cent. respectively.

The older statistical tables (Bolton, 1913¹) show for the most part the heaviest incidence of gastric ulcer (acute and chronic) between the ages of 20 and 40. In this series the highest figures are found between the ages of 40 and 60—namely, 42 per cent. of all acute ulcers and 56 per cent. of chronic.

Chronic gastric ulcer shows a slight difference in the age incidence in the two sexes, being met with rather later in life in males. This is well shown by the fact that, whereas in females only 7.5 per cent. of ulcers occur at age 60 or over, in males the proportion in these later decades is 23.5 per cent. In both sexes the heaviest incidence is in the fifth and sixth decades—52 per cent. in males, 62 per cent. in females.

In so far as chronic ulcer is concerned these figures are of only very limited value. The important point is to know the age at which gastric ulcer commences, and that, obviously, is a thing very difficult to determine. Most of the chronic ulcers in this series gave a history going back several years, many of them fifteen or twenty years. Perhaps if all the figures for chronic ulcer were moved back a decade, the result would not be far from the truth.

With regard to sex, my own figures show that chronic gastric ulcer occurs with slightly greater frequency and chronic duodenal ulcer six times more frequently in men than in women. Sir Berkeley Moynihan's (1923)²³ clinical statistics show that chronic gastric ulcer occurs with equal frequency in the two sexes, and chronic duodenal ulcer four times more frequently in men than in women.

C. MORBID ANATOMY AND HISTOLOGY.

[The lecture was illustrated with photographs of gross specimens, and with a series of histological sections mounted on lantern slides.]

On morbid anatomical grounds, both naked-eye and microscopic, it is possible to subdivide the ulcers of the stomach, and to a less extent those of the duodenum, into acute, sub-acute, and chronic varieties. The chief grounds of histological differentiation are the presence or absence of fibrosis, whether in the floor or in the margins of the ulcer, and, if present, its amount and characters. In the separation of non-inflamed acute ulcers and callous non-reacting chronic ulcers this may be almost the only distinguishing feature, but in active ulcers there are other histological criteria. An active acute ulcer in all but the earliest stages shows a zone of neutrophil polymorphic infiltration in its floor, sometimes extending a considerable way downwards from the necrotic surface. This is often accompanied by a certain amount of oedema, especially of the submucosa. It must be remembered, of course, that chronic ulcers are liable to acute exacerbations, which usually take the form of localized activity at a certain point or points of the margin or floor. Such foci will show neutrophil polymorphic reaction, but it will be superimposed on an obviously chronic lesion, and will not be so uniformly distributed as in acute ulcer.

In addition to fibrosis, chronic ulcers nearly always show foci of lymphocytic infiltration, usually in relation to small blood vessels. A certain number of plasma cells may be mixed with the lymphocytes, but these cells are not usually abundant except sometimes in the mucosa and submucosa at the margin. Infiltration of the floor of subacute and chronic ulcers by eosinophil leucocytes is common, and in a few cases reaches a high grade of intensity. Even in acute ulcers a few eosinophils may be present, but they are seldom conspicuous.

The state of the vessels in the floor of an ulcer, and beyond it, may be of help in differentiating acute and chronic. In acute ulcers the small vessels implicated in the disintegrative process are often seen to be filled with thrombi or fibrinous plugs, and even vessels of larger size may be so affected, especially if they have been ulcerated into. This is often found—for example, in cases where there is a history of haemorrhage a few days before death. In the floor of a chronic ulcer similar changes may be found, but in addition there is often well marked endarteritic and endophlebitic thickening, due to intimal proliferation. Sometimes this is associated with thrombotic obliteration of the remainder of the lumen, and sometimes with complete organization and canalization of the thrombus. Similar vascular changes are often seen on the peritoneal aspect of gastric scars.

1. Acute Ulcers.

These are usually multiple, especially in the stomach. In a series of 46 cases of acute gastric ulcer, 6 only were single. Both in stomach and duodenum they tend to be widely and irregularly distributed, with the exception that in the stomach they are usually most abundant in the pyloric half. They vary in size from tiny superficial erosions of the mucosa to large and deep ulcers measuring an inch or more in diameter. The great majority, however, are under half an inch in diameter, and most are under a quarter of an inch. In general it may be said that the more numerous the ulcers the smaller their size. I am convinced that there is no

dividing line between what have been called multiple haemorrhagic erosions and acute ulcers of larger size.

Acute ulcers are usually described as presenting a punched-out appearance, and this is equally true of both large and small varieties. As regards depth, most acute ulcers, and practically all multiple small ones, are quite shallow, confined to the mucosa, or at most the mucosa and submucosa. In the earliest stages, as seen in the *post-mortem* room, there may be almost no excavation, a quite superficial necrotic lesion with slightly reddened margins being situated on a very slightly raised and thickened patch of mucosa. Later, as the ulcer deepens, tiny vessels are exposed in the floor, which then very frequently becomes covered by a thin black layer of altered blood, or of slough infiltrated with blood. Later still, preparatory to healing, the black slough separates, leaving a clean smooth floor, in which all haemorrhage has ceased, as a result of thrombotic obliteration of the little exposed vessels. In the duodenum the slough in the floor of acute ulcers may be black and haemorrhagic, as in the stomach, but if not it tends to be bile stained. Here also there is often slight oedematous thickening around and beneath the ulcer.

Acute ulcers of larger size which are tending to penetrate the stomach wall present the well known "terraced" appearance, due to the fact that the muscular coat is penetrated over a smaller area than that occupied by the original mucosal ulcer. If the peritoneal coat should perforate, this again usually takes place over a still smaller area, and a third step is added to the terrace. The perforations in the various coats may be concentric or excentric. In my experience acute perforating ulcer of the stomach is rare. In 6,800 autopsies there were but 8 examples, of which 6 proved directly fatal—3 by perforation, 2 by haemorrhage, and 1 by mesogastric cellulitis.

Acute ulcers tend to heal readily, and if they involve only the mucosa and submucosa may do so without leaving a permanent scar. If the muscular coat is involved a certain amount of fibrosis will take place therein and a permanent scar result. In acute penetrating ulcers which heal—probably a very rare event—the permanent scar in the muscular coat will be visible from the peritoneal as well as the mucosal aspect. In a certain proportion of cases one ulcer, rarely more, fails to heal with the rest, persists, enlarges, and becomes first subacute then chronic.

2. Chronic Ulcers.

In the stomach chronic ulcers are almost invariably single. In a series of 150 cases coming to autopsy there were only 3 in which two independent chronic ulcers co-existed. It is not uncommon, on the other hand, for a chronic ulcer to be accompanied by one or more acute ulcers, a fact which is of importance in relation to the well known recurrent character of the symptoms in these cases. In the duodenum double chronic ulcer, anterior and posterior, is fairly common. In a series of 200 cases coming to autopsy there were 32 with double ulcers. Here also acute and chronic ulcers may coexist.

The position of chronic gastric ulcer is well recognized. The great majority occur along the lesser curvature or in close proximity to it, and especially in the pyloric three-fifths. On the other hand chronic ulcer is rarely seen exactly on the pylorus, and it is by no means common within an inch of it. I have analysed a series of 125 cases coming to autopsy in which the exact position of the ulcer was noted, and find that no less than 90 per cent. occur along the course of the original gastric canal.

In considering the duodenum it has been thought advisable to take both ulcers and scars into account. In 1,500 autopsies there were 74 cases with chronic duodenal lesions, all within the first part of the viscus—namely, 23 with anterior wall ulcers or scars, 18 with posterior ulcers or scars, and 33 with double lesions. In practice it was found that invariably the ulcer or scar was wholly or mainly on the anterior or posterior wall, although large posterior wall ulcers were not infrequently found to have extended on to the superior or inferior wall or both. It is in these circumstances that perforation of a posterior wall ulcer is most likely to occur.

Chronic ulcers, especially of the stomach, are usually a good deal larger than the acute variety. In a series of 40 chronic gastric ulcers 17 were under 1 inch and 8 over 3 inches in diameter. In a corresponding series of duodenal ulcers 29 were under 1 inch and 11 from 1 to 2 inches in diameter.

The naked-eyed characters of a chronic ulcer vary very much according to the state it happens to be in at the time of examination—that is, according to whether it is active, callous, or healing. As compared with acute, chronic ulcers have thickened, rounded, and overhanging margins in the active state. In callous ulcers the margins are flatter, though still often undercut, while the healing ulcer has flattened margins, with the epithelium in most cases obviously growing inwards over the floor. In the active ulcer a narrow zone of sloughing with inflammatory exudation is almost invariably present in the floor, sometimes in patches, sometimes all over. It shows up as a narrow yellow or reddish-yellow line when the fresh ulcer is cut across, and in histological preparations it stains deeply. Microscopically it consists of an intensely eosinophilic structureless groundwork, with a zone of darkly staining nuclei, or nuclear débris, in its more superficial part. In places these are obviously the nuclei of polymorph leucocytes; elsewhere they are apparently pyknotic tissue-cell nuclei. In chronic ulcers which are the seat of an acute exacerbation this necrotic or necrosing layer is very broad and irregular, and beneath it the tissues of the ulcer floor are often much infiltrated by neutrophil polymorphs. The separation of the slough is a necessary preliminary to the healing of the ulcer, as Bolton has pointed out, its presence being an effective barrier to epithelialization. Ulcers in which ingrowing of marginal epithelium can be observed have always a clean smooth floor, at least in relation to the healing part. Callous ulcers resemble healing ulcers in this respect. Either the floor is clean and fibrous, or there is a very narrow zone of necrosis without much cellular reaction.

One of the most constant features of a chronic ulcer, whether of the stomach or duodenum, is penetration of the muscular coat. I have never seen a chronic ulcer in which the muscularis was not completely transgressed, although occasionally one of subacute type may only partially involve it. Perforation is prevented mainly by the great fibrous and granulation tissue barrier formed in the floor, less often by adhesions between the stomach and some adjacent viscus—pancreas and liver especially. Pads of fat, more or less fibrosed, are commonly found in the floor of gastric and duodenal ulcers. In the stomach this is seen chiefly in lesser curvature ulcers, where the fat is merely the adipose tissue of the gastro-hepatic omentum. In the duodenum, ulcers involving the posterior wall or encroaching on the upper or lower walls show it, not anterior wall ulcers as a rule. In other cases the pad of fat may be due to adherent omentum or to retroperitoneal tissue in the lesser sac to which the ulcer has become adherent. On the other hand, it is not uncommon to find both chronic ulcers and scars totally devoid of any such fatty mass on the peritoneal aspect.

Large ulcers of great chronicity may be associated with so much inflammatory thickening in and around them that a palpable tumour may be present in the epigastrium leading to an erroneous diagnosis of cancer. Even at operation it may be difficult, sometimes impossible, to distinguish. As a rule, however, the peritoneal aspect of a simple chronic ulcer is fairly characteristic. There is thickening and opacity of the serosa as a result of fibrosis and oedema, together with vascular congestion, often intense, and not infrequently areas of haemorrhage in cases where the ulcer is active. White radiating lines of fibrosis run out from the margins of the ulcer, and there is often a vascularized fibrous band of adhesion to some adjacent structure. The floor may show fine reddish stippling owing to the presence of numerous small, thin-walled vessels in the new-formed fibrous tissue, an appearance which may be brought out at operation by gently stroking the part with gauze (Scudder). In old, completely scarred ulcers the stippling, congestion, and haemorrhage disappear and the floor of the scar becomes white and sclerotic, and may show puckering. The

shape of the ulcer crater also varies. In the stomach, in active ulcers of medium and moderately large size, it is usually deep and globular, or deep with a flat floor. Smaller ones are often similar, or may be slit-like, the mucosal margins almost meeting across the lesion. Very large ulcers, and especially callous ulcers, tend to be flat, with low, often undermined edges, and a smooth floor. Such ulcers are very frequently of the "saddle" variety, lying across, and at right angles to, the lesser curvature of the stomach. In these, as in very large posterior wall ulcers, whether gastric or duodenal, a part of the floor is usually formed by the pancreas. Healing ulcers, whether large or small, also tend to flatten out as marginal epithelialization progresses.

D. HEALING OF GASTRIC AND DUODENAL ULCER AND ITS RESULTS.

The process of healing (Stewart, 1922³¹) is similar in acute and chronic ulcers. Following the cleaning up of the floor of the ulcer—that is, the separation of sloughs or purulent material, and the disappearance of bacteria—the epithelium at the margin starts to grow inwards, while at the same time, especially in chronic ulcers, the contraction of the fibroblasts in the floor causes the ulcer to become reduced in size. Acute ulcers heal rapidly, chronic ulcers much more slowly, not only because of their larger size, but also because of the large amount of dense fibrous tissue in their floor. At the same time there can be no doubt that even very large and very chronic ulcers may heal completely, while chronic ulcers of smaller size heal with very considerable frequency. The results of healing depend on several factors, especially the size of the ulcer, its chronicity and its situation. Ulcers related to the pylorus, whether primarily gastric or duodenal, need not be of large size to produce cicatricial stenosis. In the body of the stomach ulcers of small and medium size usually heal satisfactorily; it is only large ulcers which produce serious mechanical defects (hour-glass stomach). In the duodenum, ulcers within half an inch of the pylorus frequently implicate it, and those situated lower down lead to duodenal stenosis in a small proportion of cases. In a recent series of 29 cases with completely healed scars of chronic gastric ulcers—that is, scars involving the whole thickness of the muscular wall—21, or 72 per cent., had healed satisfactorily without resulting stenosis. The figures for duodenal ulceration and scarring are similar. Even if a chronic ulcer heals completely, there is always the possibility of a new chronic ulcer making its appearance in another part of the stomach. My own statistics show that this will occur in about 9 per cent. of cases.

There are two other ways in which the recurrent character of chronic peptic ulcer may be explained. It may be due to breaking down of the old scar, a process of which it is very difficult to adduce proof, or it may be due to chronic ulcer becoming callous and quiescent, with subsequent recrudescence of ulcerative activity. Of this, both surgeons and pathologists can bring forward convincing proof.

E. COMPLICATIONS OF PEPTIC ULCER.

The more important complications of chronic gastric and duodenal ulcer are indicated in the accompanying analysis of 350 fatal cases (p. 1024).

1. Perforation.

Perforation is the most serious and one of the most common complications of both gastric and duodenal ulcer. On the other hand, the figures given in the table are obviously no index of its frequency in all cases, since the great majority of perforated ulcers are admitted because of this accident. This swells unduly the proportion of perforated cases in these statistics.

Perforation takes place rapidly in the great majority of cases by the sudden sloughing of a part or of the whole of the peritoneal floor of the ulcer, this in turn resulting from a slow, progressive devascularization. The aperture, therefore, is usually of considerable size, from one-eighth to a quarter of an inch in diameter, and it is easily seen at operation, if in an accessible position, unless concealed by

Analysis of the Causes of Death in 150 Cases of Chronic Gastric Ulcer and in 200 Cases of Chronic Duodenal Ulcer.

	Gastric.		Duodenal.	
	Total Cases.	Per cent.	Total Cases.	Per cent.
A. Causes directly attributable to the ulcer	83	55.0	145	72.5
Acute perforation ...	55	36.5	118	59.0
Subacute perforation ...	3	2.0	4	2.0
Haemorrhage ...	12	8.0	19	9.5
Carcinomatous change ...	6*	4.0	0	0.0
Effects of long-standing obstruction	4	2.5	0	0.0
Miscellaneous and doubtful ...	3	2.0	4	2.0
B. Post-operative death ...	39	25.5	20	10.0
Peritonitis ...	10	6.5	1	0.5
Haemorrhage ...	2†	1.5	1‡	0.5
Bronchopneumonia ...	10	6.5	11	5.5
Other pulmonary complications	6	4.0	2	1.0
Shock ...	3	2.0	0	0.0
Dilatation of stomach ...	2	1.5	0	0.0
Jejunal ulcer ...	0	0.0	2§	1.0
Miscellaneous and doubtful ...	6	4.0	3	1.5
C. Cause of death not related to ulcer	23	19.5	35	17.5

* Seven other ulcers showed carcinomatous change.

† Intra-gastric haemorrhage after partial gastrectomy.

‡ Haemorrhage from gastro-enterostomy operation wound.

§ One died from haemorrhage from the ulcer and one from leakage and peritonitis.

plastic exudate or adhesions. In the stomach the perforation is usually on the anterior wall and close up to the lesser curvature. In the present series of 150 cases 58 were perforated. In 81 per cent. of these the perforation was on the anterior wall, in 15.5 per cent. on the posterior, and in 3.5 per cent. on both. In the duodenum perforation is even more common. It was present in 122 cases out of 200 which came to autopsy, and in the great majority of these it was an anterior wall ulcer which had perforated. Of the anterior wall ulcers 80 per cent. had perforated, and of the posterior only 9 per cent.

The results of perforation vary with the size of the aperture and its position, and according to whether or not the stomach is empty at the time of the catastrophe. The rapid extravasation of a large quantity of stomach contents through an anterior wall perforation will result in a rapidly fatal peritonitis. The slow oozing of infected material allows time for a certain amount of limiting plastic peritonitis to occur, and the peritoneal abscesses so formed may track in various directions, up under the liver or diaphragm, including abscesses due to perforation into the lesser sac, or down into the lower abdomen or pelvis. Such localized abscesses may rupture into the general peritoneum at any stage, or, rarely, they may perforate into pleura or pericardium, through the anterior abdominal wall, or into an adjacent hollow viscus.

2. Haemorrhage.

Though much less frequent than perforation as a cause of death, this remains, in its severe form, one of the most serious complications of gastric and duodenal ulcer. In fatal cases the bleeding is generally found to originate in a chronic ulcer, and very often the open end of an artery may be seen in the floor. In the present series of 6,800 autopsies 33 deaths from ulcer were due to haemorrhage. In 13 of these the ulcer was gastric (12 chronic, 1 acute); in 20 duodenal (19 chronic, 1 acute). In the stomach it is usually a branch of the coronary artery, or the splenic or gastro-epiploic artery, which is the source of the haemorrhage, in

the duodenum the pancreatico-duodenal. In a few cases the haemorrhage comes from some adjacent viscus, liver and spleen especially, which has been penetrated by the ulcer. In the duodenum profuse haemorrhage is almost invariably due to ulcer of the posterior wall; in the stomach to ulcer of the posterior wall or lesser curvature. When bleeding is profuse there is usually haematemesis, irrespective of whether the blood is of gastric or duodenal origin, and melaena makes its appearance very soon after. If the bleeding is slight, melaena without haematemesis will result.

3. The Development of Malignant Disease.

The relation of gastric ulcer to carcinoma is one of the problems on which there is greatest diversity of opinion. Most pathologists consider that cancerous transformation of a simple chronic gastric ulcer is comparatively infrequent (Ewing, 1918,³² suggests 5 per cent. as a maximum figure), most British and American surgeons that it is a very common event (54 to 71 per cent. according to the Mayo Clinic—Mayo, 1908,³³ Wilson and McCarty, 1909³⁴). No doubt on this point *post-mortem* observations are apt to be fallacious, for the reason that the destructive effects of cancer of the stomach by the time the patient has come to autopsy may be such that the original simple ulcer has become completely destroyed. My own conclusions therefore are drawn from operation specimens only, and of these I have microscopically examined 120. The histological criterion on which ulcer-cancer cases was decided was the association in the same ulcer of an undoubted area of carcinoma with a portion which was definitely simple, and whose densely fibrous floor, devoid of cancer cells, replaced the muscularis. Suffice it to say that 10 per cent. of the cases of simple chronic ulcer coming to operation had developed cancer in the ulcer, and that in one-half of these the malignancy was unsuspected at the time of operation. Most cancerous ulcers of the stomach are, in my experience, primary cancers which have undergone central ulceration, and not simple ulcers which have become malignant.

The extraordinary rarity of duodenal carcinoma in contrast to the high ulcer incidence has long been the subject of comment, and it is often quoted as evidence against the view that chronic ulcer bears an important etiological relationship to carcinoma of the stomach. In the present series of 6,800 autopsies there were 150 cases of cancer of the stomach and only one case of primary carcinoma of the duodenum.

4. Jejunal Ulcer.

This serious and important complication of the operation of gastro-enterostomy has been of comparatively infrequent occurrence hitherto. A recent estimate by Sir Berkeley Moynihan (1923)³⁵ is that the incidence does not exceed 2 per cent., but it seems likely that further experience may show a higher figure. In the present series of 6,800 autopsies there were 16 cases in which gastro-enterostomy had been performed at periods varying from nine months to twelve years before, and in 3 of these jejunal ulceration was present. In 18 cases where an interval of from ten days to two months had elapsed since the operation, there was one case of jejunal ulceration. In each of these 4 cases the original operation had been for duodenal ulcer, and this is in keeping with clinical experience. The fact is no doubt related to the much greater degree of hyperchlorhydria present in this disease. In two of the cases death was directly attributable to the jejunal ulcer.

Jejunal ulcers may be acute or chronic, single or multiple, and they are similar in their naked-eye and microscopic characters to peptic ulcers of the stomach and duodenum. They are most common on the convexity of the jejunal loop—that is, at a point directly opposite the gastric stoma—or in the first inch or so of the efferent channel. They also occur not infrequently on the gastro-jejunal suture line—gastro-jejunal ulcer. They may cause death by perforation or haemorrhage, and apart from a fatal issue may be responsible for a return of gastric symptoms, often in an

aggravated form. Rarely they perforate into the transverse colon with resulting gastro-colic fistula.

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British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, 1923.

SECTION OF SURGERY.

Sir HENRY M. W. GRAY, K.B.E., C.B., C.M.G.,
F.R.C.S. Edin., President.

DISCUSSION ON
THE TREATMENT OF ACUTE PRIMARY
INFECTIONS OF THE HAND.

INTRODUCTORY PAPERS.

I.—D. P. D. WILKIE, O.B.E., M.Ch., F.R.C.S. Edin.,
Assistant Surgeon, Edinburgh Royal Infirmary.

THE importance of this subject, whether viewed from a medical or from an economic standpoint, can hardly be exaggerated, and I can imagine no better subject for discussion at a meeting such as this. Here we deal with the wide problems of infection, resistance, and immunity; we endeavour to crystallize our views on the principles governing our treatment of infection in both its general and local aspects, and withal, we deal with a subject of everyday importance to the practitioner of medicine. In hospital practice there is often an unfortunate tendency to regard infections of the hand as belonging to the realm of minor surgery, to be dealt with by the less experienced members of the staff; yet no cases present more difficult problems for decision, and not infrequently the life of the patient may depend on the line of treatment which is adopted. The majority of such infections are met with in the robust, wage-earning class of the community, and the difference between the rapid restoration of a hand to early and full function and the prolonged, tedious, and crippling course of the neglected or maltreated case is one of far-reaching economic import.

In the first place we must look at certain fundamental pathological considerations. The introduction of pathogenic micro-organisms into the tissues is followed by certain reactive changes, both general and local. The response by fever with its heightened metabolism, the increase in the circulating phagocytes in the blood stream, and the production of antibodies we recognize as part of Nature's effort at protection. At the seat of infection we find a reactive hyperaemia, an outpouring of lymph, and an aggregation of phagocytic cells—a local endeavour to destroy or wall off the invading germs and prevent their entrance into the blood stream. We now regard these changes as essentially protective, and we endeavour to aid and to reinforce them by such means as we possess. We know, however, that in the hand in particular the outpouring of lymph and the death of many of the phagocytes is apt to lead to an accumulation which, in certain areas, is limited as to available space, so that dangerous tension may ensue and the vascular supply and the vitality of the part may be threatened. Further, the concentrated toxins of the infecting bacteria, together with the proteolytic ferment liberated from the dead and dying phagocytes,

tend to the death and liquefaction of the surrounding tissue cells. From such an area there will be absorption of the bacterial toxins and proteoses which will have a profound and deleterious influence on the bodily economy.

The sphere of active surgical interference is the provision of drainage of such areas of suppuration and tension, and that before irreparable damage has occurred. It is essential, therefore, that we have clear ideas as to the types of infection met with, and, in the case of the suppurative variety, the sites at which pus is liable to accumulate and the routes by which it may spread.

Following on extensive inflammation, and especially on that associated with suppuration, is the inevitable fibrosis, fixing tendons, binding muscles, stiffening joints, compressing nerves and vessels, so that a limb may be helplessly crippled in every constituent part of its functional apparatus. It is essential that we bear this in mind when we are in doubt as to the necessity for what seem to be ruthless incisions. The disability following an unnecessarily extensive incision is as nothing compared to that following a burrowing, liquefying, and prolonged suppuration. Moreover, in our after-treatment it is of paramount importance to anticipate and to prevent the fixation of mobile parts by unyielding fibrous tissue, and to remember that the preservation of tissue without function is a mere waste of time and energy.

ANATOMICAL CONSIDERATIONS.

Certain points in the anatomy of the hand and digits have an important bearing on the course and spread of infection. First, the lymphatic network in the hand is very full and free, particularly on its palmar aspect, whence lymphatic trunks come to the forearm, mainly by way of the dorsum.

Secondly, the cellular tissue is divided into spaces by septa which serve to localize or guide the spread of infection. In the pulp of the digits—that is, the soft part on the palmar aspect of the terminal phalanges—dense, fibrous septa, extending down to the periosteum of the phalanx, enclose the fatty tissue, and infection of this tissue leads to great tension with necrosis, which is very apt to involve the bone. In the palm of the hand we have the deep cellular spaces so clearly demonstrated by Kanavel, of which the two most important are the middle palmar and the thenar spaces. Situated deep to the dense palmar fascia and the long tendons, and just in front of the interossei muscles, a collection of pus in the former space will point along the lumbrical muscles of the third, fourth, and fifth digits; in the thenar space it will tend to point to the radial side of the index finger. A knowledge of the existence and the boundaries of these spaces will enable us to recognize and drain a collection of pus in either or both of them before irretrievable damage has been done to the hand.

Thirdly, the tendon sheaths have an anatomical arrangement which, although not constant, is sufficiently regular and well known to require no elaboration here. An accumulation under tension in a tendon sheath will, besides endangering the vitality of the enclosed tendons, tend to rupture either into one of the palmar spaces or, in the case of the little finger and thumb—the ulnar and radial carpal bursae—into the deep cellular planes of the forearm underneath the flexor communis digitorum. In the fingers these tendon sheaths have such intimate relationship to the first interphalangeal joints, that infection of these joints is a common sequel to an infective teno-synovitis.